FILE 'CAPLUS, MEDLINE, BIOSIS,[redacted]' ENTERED AT 08:47:07 ON 04 MA[redacted]002

L1	59484	S	MULTIPLE (W) SCLEROSIS
L2	189053	S	ELISA
L3	505	S	L1 (S) L2
L4	29660	S	AUTOANTIBODY
L5	11	S	L3 (S) L4
L6	6		DUPLICATE REM L5 (5 DUPLICATES REMOVED)
L7	20488	S	MBP
L8	1868	S	L1 (S) L7
L9	42	S	L2 (S) L8
L10	760	S	ANTI (W) MBP
L11	21	S	L9 AND L10
L12	21	S	L9 (S) L10
L13	142	S	L10 (S)IGG
L14	7	S	L11 AND IGG
L15	2	S	L14 AND IGM

> d l16 1-11 ti abs so

L16 ANSWER 1 OF 11 MEDLINE DUPLICATE 1
TI An IgM **anti-MBP** Ab in a case of Waldenstrom's macroglobulinemia with polyneuropathy expressing an idiotype reactive with an MBP epitope immunodominant in MS and EAE.
AB In a previously described case of Waldenstrom's Macroglobulinemia, complicated by polyneuropathy, the IgM/lambda monoclonal antibody (mAb) was highly reactive with myelin basic protein (**MBP**). Given our demonstration that V lambda x, a recently described murine lambda variable region gene product, can itself bind **MBP** as well as confer **MBP** reactivity to an Ab, the possibility of a shared idiotypy between murine V lambda x and this human IgM/lambda **anti-MBP** was investigated. We characterized the epitope specificity of the macroglobulinemia patient's **MBP**-reactive IgM/lambda using indirect **ELISA** procedures with **MBP**, a citrullinated isomer of **MBP** termed C8, or peptide fragments of **MBP** as the coating antigens and monospecific Ab to V lambda x as the secondary Ab. The patient's **MBP**-reactive IgM/lambda was recognized by Ab specific for V lambda x and, like murine mAb containing V lambda x bound human **MBP** but not **MBP**-C8 nor other common autoantigens such as DNA, thyroglobulin, or actin. The **anti-MBP** reactivity was selective for **MBP** peptide 90-170 and preferentially recognized **MBP** peptide 84-96. Thus, the patient's macroglobulin and perhaps certain other human Ab with a 'V lambda x idiotype' bind to **MBP** peptide residues 84-96, an immunodominant peptide in **multiple sclerosis** patients. Such binding may be involved in the pathogenesis of neural damage in patients with neuroimmunologic disorders related to plasma cell dyscrasias or autoimmunity.
SO JOURNAL OF NEUROIMMUNOLOGY, (2001 Feb 1) 113 (1) 163-9.
Journal code: HSO. ISSN: 0165-5728.

L16 ANSWER 2 OF 11 MEDLINE DUPLICATE 2
TI Detection of brain-specific autoantibodies to myelin oligodendrocyte glycoprotein, S100beta and myelin basic protein in patients with Devic's neuromyelitis optica.
AB Neuromyelitis optica (NMO) is a rare syndrome characterized by the combination of acute optic neuritis and transverse myelitis, usually not seen in **Multiple Sclerosis** (MS) and other demyelinating syndromes of the central nervous system (CNS). A high prevalence of various autoantibodies has been described in patients with NMO suggesting a polyclonal activation of the humoral immune system. We examined autoantibody responses to myelin (**MBP**, MOG with isotypes and epitopes) and astroglial (S100beta) antigens in four patients with NMO by **ELISA** and Immunoblot. All patients showed a positive anti-MOG response, with one showing reaction to the MOG epitope corresponding to amino acid sequence 63-87. **MBP**-autoantibodies were only detected in two and S100beta-autoantibodies in one patient. Despite the limited number of samples, these findings suggest a predominant anti-MOG rather than **anti-MBP** or anti-S100beta autoantibody response in NMO, though no NMO-specific antibody pattern was found, which is in keeping with a widespread acute immune activation, including a strong B-cell response.
SO NEUROSCIENCE LETTERS, (2001 Jul 13) 307 (2) 131-3.
Journal code: N7N; 7600130. ISSN: 0304-3940.

L16 ANSWER 3 OF 11 MEDLINE DUPLICATE 3
TI Clinical and analytical evaluation of an enzyme immunoassay for myelin basic protein in cerebrospinal fluid.
AB BACKGROUND: RIA of myelin basic protein (**MBP**) in cerebrospinal fluid (CSF) is commonly used as a biochemical marker of demyelination in patients with **multiple sclerosis** (MS). Our aim was to develop a sufficiently sensitive **ELISA** for **MBP** and evaluate it clinically in patients with MS. METHODS: The **ELISA** used anti-bovine **MBP** antibody coated on plates and biotinylated **anti-MBP** antibody. The bound antibody complex was

quantified with streptavid[redacted]horseradish peroxidase. **MBP** wa[redacted] determined in CSF from 84 MS patients and 55 patients with other neurological diseases. RESULTS: The respective within- and between-assay CVs were 4.7% and 7.2% at 200 ng/L, and 6. 3% and 8.8% at 2000 ng/L. The detection limit was 30 ng/L. Most of the MS patients with acute exacerbations had markedly increased **MBP** in the CSF. Longitudinal studies of six MS patients with recurrent exacerbation confirmed this observation. **MBP** concentrations from 78 MS patients, as tested with our **ELISA**, correlated well with those obtained by RIA (r = 0.9; P: <0.01), but the detection limit of the **ELISA** was much lower than that of the RIA. CONCLUSIONS: This convenient **ELISA** with higher sensitivity than the existing assays is a suitable routine assay that provides a diagnostic indicator of myelin breakdown in the central nervous system; moreover, it is an excellent indicator of MS disease activity.

SO CLINICAL CHEMISTRY, (2000 Sep) 46 (9) 1326-30.
Journal code: DBZ; 9421549. ISSN: 0009-9147.

L16 ANSWER 4 OF 11 MEDLINE DUPLICATE 4

TI High levels of cerebrospinal fluid IgM binding to myelin basic protein are associated with early benign course in multiple sclerosis.

AB We assessed human myelin basic protein (**MBP**) binding IgM levels in CSF. **MBP** is the most studied putative antigen in **multiple sclerosis** (MS) and immune responses against it may be involved in the demyelination process. We also correlated these levels with EDSS score and other parameters of disease progression and prognosis, both at the time of CSF analysis and during follow-up. CSF IgM **anti-MBP** levels were assayed by measuring total IgM levels with solid-phase **ELISA** in CSF samples from 66 patients with relapsing-remitting MS, 11 subjects without neurological diseases, 20 patients with non-inflammatory neurological diseases and 7 patients with lymphocytic meningitis, before and after immunoabsorption with human **MBP**. Confirmation of IgM binding specificity was performed by immunoblotting of positive CSF samples onto **MBP** coated-nitrocellulose sheets. Clinical evaluation (disability score, number and time of attacks) was performed during a mean follow-up of 2.7 +/- 1.1 years. 23 of the 66 relapsing-remitting MS patients (33.8%) had elevated IgM **anti-MBP** levels. In this patient subgroup, IgM **anti-MBP** levels correlated with the IgM index (r = 0.71; P = 0.0001), but not with CSF/serum albumin (r = 0.08; P = 0.72). In the first year of follow-up, patients with low IgM **anti-MBP** suffered from more numerous attacks than those with elevated levels (0.86 +/- 0.63 versus 0.43 +/- 0.58; P = 0.017). Patients with high IgM binding to **MBP** had a first attack during follow-up in a significantly higher time than those with low binding (28.87 +/- 4.7 versus 17 +/- 2.6 months, respectively; P = 0.005) and reached a decrease of 0.5 EDSS point significantly faster than those with low IgM (16.17 +/- 1.2 versus 29.7 +/- 2.6 months, respectively; P = 0.0002). A similar significant finding was observed when the time to reach low disability score (EDSS < or = 2.0) was analyzed (10.7 +/- 2.57 +/- 3.3 months, respectively; P = 0.014). These findings demonstrate that in a subgroup of MS patients, elevated CSF levels of IgM **anti-MBP** are associated with early favorable course and therefore suggest that IgM binding to **MBP** could be a possible prognostic marker in relapsing-remitting MS to select early MS patients for future trials.

SO JOURNAL OF NEUROIMMUNOLOGY, (1997 Jul) 77 (1) 128-33.
Journal code: HSO; 8109498. ISSN: 0165-5728.

L16 ANSWER 5 OF 11 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 5

TI ELISA for myelin basic protein and **anti-MBP** in serum and cerebral spinal fluid of patients with disease of the nervous system

AB Myelin basic protein (**MBP**) and antibodies to myelin basic protein (**Anti-MBP**) were used as biochem. indicators in identification of the presence of acute brain injury and demyelination neuropathy, and were tested by a simplified **ELISA** on 337 patients with diseases of the nervous system including 36 compressive

disease (CMP), 33 **multiple sclerosis** (MS), 34 cerebrovascular diseases (CVD), 31 inflammatory diseases of central nervous system (ID), 161 epilepsy (EP) and 42 other nervous diseases (OND). The serum mean MBP values of CMP, MS, CVD, ID and EP groups were significantly higher than those of OND group and normal control ($P < 0.01$). The serum mean MBP value of 33 acute trauma patients with spine fracture and paraplegia (the majority of CMP group) was the highest as compared with MS ($P < 0.05$), CVD, ID and EP groups ($P < 0.01$). CSF mean MBP value of 15 CVD patients was markedly greater than that of OND group ($P < 0.05$). No statistically significant differences were found in serum MBP values between OND group and normal control and in serum and CSF **Anti-MBP** values among 6 groups.

SO Huaxi Yike Daxue Xuebao (1995), 26(2), 131-4
CODEN: HYDXET; ISSN: 0257-7712

L16 ANSWER 6 OF 11 MEDLINE

TI Lead alters the immunogenicity of two neural proteins: a potential mechanism for the progression of lead-induced neurotoxicity.

AB Some heavy metals have been suspected of playing a role in the pathogenesis of nervous system diseases such as **multiple sclerosis**, amyotrophic lateral sclerosis, and Alzheimer's disease. In these disorders, autoantibodies against neural proteins are evident at some stage of the disease. Lead is known to affect both the immune and nervous systems. Work in our laboratory has shown that lead exposure leads to the production of autoantibodies against neural proteins, including myelin basic protein (**MBP**) and glial fibrillary acidic protein (GFAP). We hypothesize that lead aggravates neurological disease by enhancing the immunogenicity of nervous system proteins, including **MBP** and GFAP. To test this hypothesis, lead-altered protein was prepared by incubating **MBP** or GFAP with lead acetate for 24 hr. On days 0, 14, and 28, mice received inoculations with either saline, native protein, or lead-altered protein. **Anti-MBP** and anti-GFAP, isotypes IgM and IgG, were measured in sera by **ELISA** on day 38. Sera of mice treated with lead-altered **MBP** had statistically higher **anti-MBP** IgG titers than both control and native **MBP**-immunized mice. An analogous response was seen in mice immunized with lead-altered GFAP. Supernatants from lectin-stimulated splenocytes were also examined for antibody titers and for interleukin 2 (IL-2) and interleukin 6 (IL-6) levels. A significant increase in IL-6 production was seen in mice immunized with lead-altered **MBP** but not with lead-altered GFAP. No changes were observed in the IL-2 levels of mice immunized with either lead-altered protein.(ABSTRACT TRUNCATED AT 250 WORDS)

SO ENVIRONMENTAL HEALTH PERSPECTIVES, (1994 Dec) 102 (12) 1052-6.
Journal code: EI0; 0330411. ISSN: 0091-6765.

L16 ANSWER 7 OF 11 MEDLINE

TI [Determination of IgG subclass antibodies to the basic myelin protein in patients with multiple sclerosis].
Badania nad okresleniem podklas IgG przeciwcial przeciwko bialku zasadowemu mieliny u pacjentow ze stwardnieniem rozsianym.

AB The aim of the study was to determine IgG subclasses of **MBP** antibodies in patients with **multiple sclerosis**. The enzyme-linked immunosorbent assay (**ELISA**) with monoclonal antibodies against human IgG subclasses was applied. **Anti-MBP** antibodies were found in the CSF of 16% and in the sera of 20% patients with MS. **MBP** antibodies belonged to IgG1, IgG2 and IgG3 subclasses (in the CSF) and to IgG1 and IgG3 subclasses (in the sera). The role of **MBP** antibodies in context of their IgG subclasses distribution is discussed.

SO NEUROLOGIA I NEUROCHIRURGIA POLSKA, (1993 Nov-Dec) 27 (6) 803-9.
Journal code: NYF; 0101265. ISSN: 0028-3843.

L16 ANSWER 8 OF 11 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

TI Detection of **anti-MBP** in the serum of patients with multiple sclerosis.

AB The pathogenesis of **multiple sclerosis** (M.S.) probably

involves immune response against nervous tissue antigens, possibility antibodies directed against myelin basic protein (**anti-MBP**). Aim of this study was to investigate **anti-MBP** in the serum of patients with M.S. since the occurrence of these antibodies in subjects with M.S. is controversial. We used enzyme-linked immunosorbent assay (**ELISA**) plates, coated with porcine **MBP** to determine **anti-MBP** of the IgG, IgA, IgM isotypes in serum of 20 patients with M.S. and control group 44 patients with neurosis or nonspecific low back pain. Twelve (60%) of M.S. serum samples revealed **anti-MBP** antibodies levels exceeding 3SD of the control group. Six of the 12 patients with high levels **anti-MBP** in serum had **anti-MBP** IgG class, 2 IgA class, 2 IgG and IgA classes and 2 antibodies of three isotypes.

SO Deltion Ellenikes Mikrobiologikes Etaireias, (1992) Vol. 37, No. 6, pp. 667-672.
ISSN: 0438-9573.

L16 ANSWER 9 OF 11 MEDLINE DUPLICATE 6

TI An immunochemical comparison of human myelin basic protein and its modified, citrullinated form, C8.

AB An immunochemical analysis was conducted to compare the C1 isomer of human myelin basic protein (**MBP**) with the newly described and less cationic, citrullinated isomer of **MBP** referred to as C8. Ten polyclonal antisera directed at multiple epitopes or restricted regions of **MBP** were used in radioimmunoassays to examine **MBP**-C1 and **MBP**-C8. Antisera reactive with **MBP** peptide 1-14 clearly distinguished **MBP**-C1 from **MBP**-C8. Antisera to human **MBP** peptides 10-19 and 90-170, but not to **MBP** peptide 69-89, showed modest differences between **MBP**-C1 and **MBP** -C8. The **MBP**-C8s from **multiple sclerosis** (MS) and non-MS brain reacted essentially the same. With murine monoclonal antibodies and enzyme-linked immunosorbent assay (**ELISA**), differences between **MBP**-C8 and other isomers were shown for **anti-MBP** 10-19 but not for **anti-MBP** 1-9 or **anti-MBP** 80-89. These findings imply differences in sequence or conformation in the structure of **MBP** -C7 compared to **MBP**-C1, most notably near the amino terminus.

SO JOURNAL OF NEUROIMMUNOLOGY, (1992 Feb) 36 (2-3) 135-46.
Journal code: HSO; 8109498. ISSN: 0165-5728.

L16 ANSWER 10 OF 11 MEDLINE DUPLICATE 7

TI Immunoblot detection of oligoclonal anti-myelin basic protein IgG antibodies in cerebrospinal fluid in multiple sclerosis.

AB Migration properties and occurrence of antibodies against myelin basic protein (**MBP**) in paired CSF and serum specimens from patients with **multiple sclerosis** (MS) were demonstrated after agarose isoelectric focusing, immunoblot transfer, and immunoperoxidase staining. Oligoclonal IgG antibody bands directed against **MBP** were found in the CSF of 9 of 28 patients with MS (32%), but not in the CSF of any of 34 patients with other neurologic diseases. No serum showed **anti-MBP** antibody bands. The CSF **anti-MBP** antibodies migrated to the anodal region of the IgG area in a different fashion from oligoclonal IgG and anti-measles IgG antibodies, which were detected in parallel. The **anti-MBP** bands were transient in three of seven patients whom we studied consecutively. Enzyme-linked immunosorbent assay (**ELISA**) of serum and CSF for detection of IgG reactivity against **MBP** showed absorbance values above 2 standard deviations of controls in 44% of the MS patients and in 21% of those with other neurologic diseases. Results of this assay correlated partly with those of the immunoblot assay. **ELISA** positive and immunoblot negative results might be due to a broad polyclonal **anti-MBP** antibody response.

SO NEUROLOGY, (1987 Sep) 37 (9) 1515-9.
Journal code: NZ0; 0401060. ISSN: 0028-3878.

L16 ANSWER 11 OF 11 MEDLINE DUPLICATE 8

TI Serum and cerebrospinal fl[redacted] antibodies against myelin bas[redacted] protein and their IgG subclass distribution in multiple sclerosis.

AB IgG class antibodies reactive with myelin basic protein (**MBP**) were determined by enzyme-linked immunosorbent assay (**ELISA**) in serum and cerebrospinal fluid (CSF) of 37 patients with **multiple sclerosis** and a control group of 32 patients with tension headache or psychoneurosis. Using standardised amounts of IgG from CSF and serum in **ELISA**, significantly higher mean antibody levels were found in CSF as well as in serum from the patients with **multiple sclerosis**. Ten (27%) of the **multiple sclerosis** CSF samples and 15 (41%) of the **multiple sclerosis** sera revealed **anti MBP** antibody levels exceeding 2 SD of the control group. Seven patients (19%) showed exclusive or higher levels of **anti MBP** antibodies in CSF, suggesting synthesis within the central nervous system. Analysis by **ELISA** for IgG subclasses of **anti MBP** antibodies revealed that they were restricted to IgG 1 in four patients and IgG 3 in one.

SO JOURNAL OF NEUROLOGY, NEUROSURGERY AND PSYCHIATRY, (1986 Sep) 49 (9) 1066-70.
Journal code: JBB; 2985191R. ISSN: 0022-3050.

=> d l16

L16 ANSWER 1 OF 11 MEDLINE DUPLICATE 1
AN 2001108741 MEDLINE
DN 21065710 PubMed ID: 11137588
TI An IgM **anti-MBP** Ab in a case of Waldenstrom's macroglobulinemia with polyneuropathy expressing an idiotype reactive with an MBP epitope immunodominant in MS and EAE.
AU Noerager B D; Inuzuka T; Kira J; Blalock J E; Whitaker J N; Galin F S
CS Department of Physiology and Biophysics, University of Alabama at Birmingham, Birmingham, AL 35294, USA.
NC AI37670 (NIAID)
NS29719 (NINDS)
SO JOURNAL OF NEUROIMMUNOLOGY, (2001 Feb 1) 113 (1) 163-9.
Journal code: HSO. ISSN: 0165-5728.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200102
ED Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010208

=> FIL STNGUIDE

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=> d l16 11
YOU HAVE REQUESTED DATA FROM FILE 'CAPLUS, MEDLINE, BIOSIS' - CONTINUE? (Y)/N:y

6 ANSWER 5 OF 11 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 5

TI ELISA for myelin basic protein and **anti-MBP** in serum and cerebral spinal fluid of patients with disease of the nervous system

AB Myelin basic protein (**MBP**) and antibodies to myelin basic protein (**Anti-MBP**) were used as biochem. indicators in identification of the presence of acute brain injury and demyelination neuropathy, and were tested by a simplified **ELISA** on 337 patients with diseases of the nervous system including 36 compressive disease (CMP), 33 **multiple sclerosis** (MS), 34 cerebrovascular diseases (CVD), 31 inflammatory diseases of central nervous system (ID), 161 epilepsy (EP) and 42 other nervous diseases (OND). The serum mean MBP values of CMP, MS, CVD, ID and EP groups were significantly higher than those of OND group and normal control ($P < 0.01$). The serum mean MBP value of 33 acute trauma patients with spine fracture and paraplegia (the majority of CMP group) was the highest as compared with MS ($P < 0.05$), CVD, ID and EP groups ($P < 0.01$). CSF mean MBP value of 15 CVD patients was markedly greater than that of OND group ($P < 0.05$). No statistically significant differences were found in serum MBP values between OND group and normal control and in serum and CSF **Anti-MBP** values among 6 groups.

SO Huaxi Yike Daxue Xuebao (1995), 26(2), 131-4
CODEN: HYDXET; ISSN: 0257-7712

L16 ANSWER 11 OF 11 MEDLIN[redacted] DUPLICAT[redacted]

TI Serum and cerebrospinal fluid antibodies against myelin basic protein and their IgG subclass distribution in multiple sclerosis.

AB IgG class antibodies reactive with myelin basic protein (**MBP**) were determined by enzyme-linked immunosorbent assay (**ELISA**) in serum and cerebrospinal fluid (CSF) of 37 patients with **multiple sclerosis** and a control group of 32 patients with tension headache or psychoneurosis. Using standardised amounts of IgG from CSF and serum in **ELISA**, significantly higher mean antibody levels were found in CSF as well as in serum from the patients with **multiple sclerosis**. Ten (27%) of the **multiple sclerosis** CSF samples and 15 (41%) of the **multiple sclerosis** sera revealed **anti MBP** antibody levels exceeding 2 SD of the control group. Seven patients (19%) showed exclusive or higher levels of **anti MBP** antibodies in CSF, suggesting synthesis within the central nervous system. Analysis by **ELISA** for IgG subclasses of **anti MBP** antibodies revealed that they were restricted to IgG 1 in four patients and IgG 3 in one.

SO JOURNAL OF NEUROLOGY, NEUROSURGERY AND PSYCHIATRY, (1986 Sep) 49 (9) 1066-70.
Journal code: JBB; 2985191R. ISSN: 0022-3050.

L16 ANSWER 7 OF 11 MEDLINE

TI [Determination of IgG subclass antibodies to the basic myelin protein in patients with multiple sclerosis].
Badania nad okresleniem podklas IgG przeciwcial przeciwko bialku zasadowemu mieliny u pacjentow ze stwardnieniem rozsianym.

AB The aim of the study was to determine IgG subclasses of **MBP** antibodies in patients with **multiple sclerosis**. The enzyme-linked immunosorbent assay (**ELISA**) with monoclonal antibodies against human IgG subclasses was applied. **Anti-MBP** antibodies were found in the CSF of 16% and in the sera of 20% patients with MS. **MBP** antibodies belonged to IgG1, IgG2 and IgG3 subclasses (in the CSF) and to IgG1 and IgG3 subclasses (in the sera). The role of **MBP** antibodies in context of their IgG subclasses distribution is discussed.

SO NEUROLOGIA I NEUROCHIRURGIA POLSKA, (1993 Nov-Dec) 27 (6) 803-9.
Journal code: NYF; 0101265. ISSN: 0028-3843.

not in Lib

d l6 1-6 ti abs so

L6 ANSWER 1 OF 6 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI Protein array characterization of the specificity of the autoantibody response in experimental autoimmune encephalomyelitis: Examination of the role of epitope spreading in disease progression.
AB Experimental autoimmune encephalomyelitis (EAE) is an autoimmune demyelinating disease of the central nervous system, and serves as an animal model for **multiple sclerosis** (MS). EAE results from induction of an autoimmune response directed against the protein components of the myelin sheath. Protein arrays contain thousands of different proteins and/or peptides applied to the surface of microscope slides where they can be analyzed for their interactions with autoantibodies in disease and control samples. Protein array technology was developed by Patrick Brown and others, and we have refined protein array technology to perform large-scale profiling of the specificity of the **autoantibody** response in individuals. We have developed a MS/EAE 'myelin proteome' array that contains 400 distinct protein and peptides derived from and representing protein components of the myelin sheath. Using this 'myelin proteome' array we demonstrate that following induction of EAE in SJL mice with a single peptide (PLPp139-151), the **autoantibody** response spreads to involve multiple epitopes on 3 additional myelin proteins (MOG, MBP, and CNPase). We observe increased spreading of the autoimmune response in mice that develop, compared to mice that do not develop, relapsing EAE. Mice treated for EAE by tolerizing vaccination with DNA encoding myelin proteins (see abstract by Garren, et al.) had reduced spreading of the **autoantibody** response. We have correlated our protein array data with **ELISA** and T cell proliferation assay results. Our observation that increased epitope spreading of the **autoantibody** response is associated with relapsing disease has important implications regarding the mechanisms underlying autoimmune disease progression.
SO FASEB Journal, (March 8, 2001) Vol. 15, No. 5, pp. A1064. print.
Meeting Info.: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001
ISSN: 0892-6638.

L6 ANSWER 2 OF 6 MEDLINE DUPLICATE 1
TI Detection of brain-specific autoantibodies to myelin oligodendrocyte glycoprotein, S100beta and myelin basic protein in patients with Devic's neuromyelitis optica.
AB Neuromyelitis optica (NMO) is a rare syndrome characterized by the combination of acute optic neuritis and transverse myelitis, usually not seen in **Multiple Sclerosis** (MS) and other demyelinating syndromes of the central nervous system (CNS). A high prevalence of various autoantibodies has been described in patients with NMO suggesting a polyclonal activation of the humoral immune system. We examined **autoantibody** responses to myelin (MBP, MOG with isotypes and epitopes) and astroglial (S100beta) antigens in four patients with NMO by **ELISA** and Immunoblot. All patients showed a positive anti-MOG response, with one showing reaction to the MOG epitope corresponding to amino acid sequence 63-87. MBP-autoantibodies were only detected in two and S100beta-autoantibodies in one patient. Despite the limited number of samples, these findings suggest a predominant anti-MOG rather than anti-MBP or anti-S100beta **autoantibody** response in NMO, though no NMO-specific antibody pattern was found, which is in keeping with a widespread acute immune activation, including a strong B-cell response.
SO NEUROSCIENCE LETTERS, (2001 Jul 13) 307 (2) 131-3.
Journal code: N7N; 7600130. ISSN: 0304-3940.

L6 ANSWER 3 OF 6 MEDLINE DUPLICATE 2
TI The N-terminal domain of the myelin oligodendrocyte glycoprotein (MOG) induces acute demyelinating experimental autoimmune encephalomyelitis in the Lewis rat.
AB Using a highly purified recombinant protein, mMOG, we demonstrated that

autoimmune responses to th[redacted]-terminal domain (a.a 1-125) o[redacted]he myelin oligodendrocyte glycoprotein (MOG) induce an acute demyelinating variant of experimental autoimmune encephalomyelitis (EAE) in the Lewis rat. Immunisation with 100 micrograms of mMOG in adjuvant at the base of the tail induced mild clinical disease in 9 of 11 animals (mean clinical score 1.1). The disease was characterised histopathologically by the presence of inflammation and focal demyelinating lesions in the central nervous system (CNS). Adoptive transfer experiments suggest that this inflammatory demyelinating pathology is mediated by synergy between a weakly encephalitogenic, MOG-specific T cell response and a demyelinating, MOG-specific **autoantibody** response. Using in vitro selected mMOG-reactive T cell lines, the encephalitogenic T cell response to this domain of MOG was found to recognise two distinct epitopes, MOG1-20 and MOG35-55; whereas **ELISA** demonstrated that the immunodominant B cell epitope was located within the amino acid sequence MOG1-25. However although active immunisation with synthetic peptides corresponding to the T cell epitopes, MOG1-20 or MOG35-55, induced an inflammatory response in the CNS, this was not associated with demyelination indicating that the demyelinating antibody response recognises other, possibly conformation dependent epitopes. This study unequivocally demonstrates that MOG-specific autoimmune responses are alone sufficient to induce a demyelinating disease of the CNS and supports the proposal that MOG may play an important role in the immunopathogenesis of **multiple sclerosis**.

SO JOURNAL OF NEUROIMMUNOLOGY, (1995 Dec) 63 (1) 17-27.
Journal code: HSO; 8109498. ISSN: 0165-5728.

L6 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 3
TI ELISA-type titertray assay for IgM anti-GM1 autoantibodies
AB The authors report an ELISA-type titertray assay for autoantibodies against the ganglioside GM1. Trays were coated with ganglioside GM1 and reacted with patients' sera; bound IgM was detected with rabbit antibody to human IgM. Higher-titer serum from a patient was used as calibrator, another patient's serum as the pos. control, and the GM1-specific cholera toxin as the control for GM1 coating. Regression curves of serum titers obtained from different patients were linear and parallel. Intra- and inter-assay CVs were 4.0-7.8% and 5.5-16%, resp. The authors detected antibodies at a titer of 1:250 in normal subjects. Anal. specificity of the calibrator serum against GM1 was demonstrated by immune thin-layer chromatog. Anti-GM1 antibodies were increased in patients with chronic inflammatory demyelinating polyradiculoneuropathy or multiple sclerosis. In Guillain-Barre syndrome, preliminary longitudinal studies showed a decrease in anti-GM1 titer that was related to clin. recovery.
SO Clin. Chem. (Washington, D. C.) (1994), 40(7, Pt. 1), 1331-4
CODEN: CLCHAU; ISSN: 0009-9147

L6 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 4
TI Means and methods for in vitro diagnosis of multiple sclerosis and other demyelinating neuropathies using inositol group antigens
AB Multiple sclerosis and other demyelinating diseases are diagnosed by using the inositol group as antigenic determinant. Kits for the assay comprise inositol-contg. antigens and solvents, buffers, and agents necessary for carrying out the assay. An ELISA was used to detect anti-phosphatidylinositol autoantibodies in the blood serum of multiple sclerosis patients.
SO Fr. Demande, 29 pp.
CODEN: FRXXBL

L6 ANSWER 6 OF 6 MEDLINE DUPLICATE 5
TI Human monoclonal autoantibodies produced by hybridomas derived from lymphocytes of multiple sclerosis patients.
AB The aim of this study was to characterize autoantibodies produced in vitro by peripheral blood lymphocytes (PBL) of patients affected with **multiple sclerosis** (MS). We studied supernatants from man-mouse hybridomas established by fusion of PBL from 6 MS patients (group I) and from 13 individuals free of any neurological pathology (group II) with the mouse myeloma cell line P3X63 Ag8-653. They were

screened for human IgG or [redacted] production by **ELISA**. **Autoantibody** activity against lymphocytes was studied by cell-binding **ELISA**. Anti-tissue reactivity was assessed by indirect immunofluorescence assay (IFA) on human cerebellum and peripheral nerve as well as on a panel of 8 non-nervous tissues. Additional **ELISA** tests were performed on 4 purified cellular antigens. Among 522 supernatants in group I, 13.7% contained Ig, mainly IgM, as compared to 25% among 1212 supernatants in group II; 8.3% in group I and 6.7% in group II contained anti-tissue autoantibodies. Antibodies against purified cellular antigens were found in 6% of the supernatants in group II versus 7% in group II. One human monoclonal anti-astrocyte antibody from group I was further studied. This IgM lambda (SAN-7) was particularly polyreactive and recognized glial fibrillar acid protein and other intermediate filaments, as well as tubulin and myosin. Moreover, cross-reactivity was observed with a hapten (TNP-BSA).

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	Type	L #	Hits	Search Text	DBs	Time Stamp	Comments
1	BRS	L1	1440	autoantibod$	USPAT; US-PGPUB	2002/03/04 14:52	
2	BRS	L2	5799	multiple adj sclerosis	USPAT; US-PGPUB	2002/03/04 14:52	
3	BRS	L3	101	1 same 2	USPAT; US-PGPUB	2002/03/04 14:52	
4	BRS	L4	1151	myelin adj basic	USPAT; US-PGPUB	2002/03/04 14:52	
5	BRS	L5	9	3 same 4	USPAT; US-PGPUB	2002/03/04 14:52	

	Type	L #	Hits	Search Text	DBs	Time Stamp	Comments
1	BRS	L1	2356777	diagnos$ or monitor or predict or determin$	USPAT; US-PGPUB; EPO; DERWENT	2002/03/04 11:10	
2	BRS	L2	11331	multiple adj sclerosis	USPAT; US-PGPUB; EPO; DERWENT	2002/03/04 11:10	
3	BRS	L3	2004	1 same 2	USPAT; US-PGPUB; EPO; DERWENT	2002/03/04 11:10	
4	BRS	L4	671	3 and elisa	USPAT; US-PGPUB; EPO; DERWENT	2002/03/04 11:11	
5	BRS	L5	7188	enzyme adj linked adj immunosorbent adj assay	USPAT; US-PGPUB; EPO; DERWENT	2002/03/04 11:12	
6	BRS	L6	463	3 and 5	USPAT; US-PGPUB; EPO; DERWENT	2002/03/04 11:12	

	Type	L #	Hits	Search Text	DBs	Time Stamp	Comments
7	BRS	L7	59	4 and autoantibod$	USPAT; US-PGPUB; EPO; DERWENT	2002/03/04 11:13	
8	BRS	L8	7781	(myelin adj basic adj protein)or mbp	USPAT; US-PGPUB; EPO; DERWENT	2002/03/04 11:14	
9	BRS	L10	115	anti adj mbp	USPAT; US-PGPUB; EPO; DERWENT	2002/03/04 11:14	
10	BRS	L11	6	9 and 10	USPAT; US-PGPUB; EPO; DERWENT	2002/03/04 11:15	
11	BRS	L9	29	7 and 8	USPAT; US-PGPUB; EPO; DERWENT	2002/03/04 11:18	

	Type	L #	Hits	Search Text	DBs	Time Stamp	Comments
1	BRS	L1	403131	diagnos$ or monitor or predict$	USPAT; US-PGPUB	2002/03/04 10:10	
2	BRS	L2	5799	multiple adj sclerosis	USPAT; US-PGPUB	2002/03/04 10:11	
3	BRS	L3	801	1 same 2	USPAT; US-PGPUB	2002/03/04 10:12	
4	BRS	L4	253167	blood or serum or plasma	USPAT; US-PGPUB	2002/03/04 10:13	
5	BRS	L5	757	3 and 4	USPAT; US-PGPUB	2002/03/04 10:13	
6	BRS	L6	1440	autoantibod$	USPAT; US-PGPUB	2002/03/04 10:14	
7	BRS	L7	44	5 and 6	USPAT; US-PGPUB	2002/03/04 10:14	
8	BRS	L9	6603	enzyme adj linked adj immunosorbent adj assay	USPAT; US-PGPUB	2002/03/04 10:15	
9	BRS	L10	26	7 and 9	USPAT; US-PGPUB	2002/03/04 10:15	
10	BRS	L8	35	7 and elisa	USPAT; US-PGPUB	2002/03/04 10:49	
11	BRS	L11	35	7 and elisa	USPAT; US-PGPUB; EPO; DERWENT	2002/03/04 10:50	